

U.S. Department of Labor

Office of Administrative Law Judges
800 K Street, NW, Suite 400-N
Washington, DC 20001-8002

(202) 693-7300
(202) 693-7365 (FAX)



Issue Date: 26 July 2005

In the Matter of:

MOLLY SEXTON, widow of
JAMES G. SEXTON
Claimant

Case No.: 2004 BLA 76

v.

PARAMOUNT COAL CO.,
Employer

and

DIRECTOR, OFFICE OF WORKERS'
COMPENSATION PROGRAMS
Party in Interest

Appearances:

Mr. Joseph E. Wolfe, Attorney
For the Claimant

Mr. Timothy W. Gresham, Attorney
For the Employer

Before:

Richard T. Stansell-Gamm
Administrative Law Judge

DECISION AND ORDER – AWARD OF SURVIVOR BENEFITS

This matter involves a claim filed by Mrs. Molly Sexton for benefits under the Black Lung Benefits Act, Title 30, United States Code, Sections 901 to 945 (“the Act”). Benefits are awarded to persons who are totally disabled within the meaning of the Act due to pneumoconiosis, or to survivors of persons who died due to pneumoconiosis. Pneumoconiosis is a dust disease of the lung arising from coal mine employment and is commonly known as “black lung” disease.

Procedural Background

Initial Claim

After her husband's death, Mrs. Molly Sexton filed her claim for survivor benefits on February 23, 2000 (DX 1).¹ Following a preliminary review of the medical evidence in the record, the District Director indicated that Mrs. Sexton would be entitled to benefits on May 5, 2000 (DX 27). The Employer objected to the preliminary findings on June 1, 2000 (DX 29). Upon review of additional evidence, the Director determined that Mrs. Sexton was entitled to benefits on August 7, 2000 (DX 33). On August 17, 2000, the Employer appealed the determination (DX 34). As a result, the Black Lung Trust Fund initiated interim benefits to Mrs. Sexton on August 25, 2000 (DX 35), and the case was forwarded to the Office of Administrative Law Judges ("OALJ") on September 6, 2000 for a hearing (DX 36).

Administrative Law Judge Edward Terhune Miller conducted a hearing on January 8, 2001 (DX 47). On October 31, 2001, Judge Miller issued a decision awarding Ms. Sexton survivor benefits, finding that the presence of complicated pneumoconiosis invoked the presumption that Mr. Sexton's death was due to pneumoconiosis (DX 52). Employer appealed the award of benefits to the Benefits Review Board ("BRB" or "Board") (DX 53). On October 30, 2002, the BRB reversed Judge Miller's decision. The Board determined that in the absence of supportive medical opinion Judge Miller's conclusion that a two centimeter mass discovered during an autopsy was equivalent to a radiographic lesion greater than one centimeter mass was legally insufficient to support his finding that Mr. Sexton had complicated pneumoconiosis (DX 69).²

Modification Request

Pursuant to a letter written on August 21, 2003, counsel for Mrs. Sexton requested modification of the prior final determination by the Board (DX 70). Since no medical evidence was submitted with her modification request, Mrs. Sexton's request was forwarded to the OALJ on January 27, 2004 for a determination of whether a mistake of fact was present in the Board's denial of her claim (DX 72 and DX 74). Pursuant to a Notice of Hearing dated March 18, 2004 (ALJ 1), I conducted a hearing in this case on June 10, 2004 in Abingdon, Virginia, attended by Mrs. Sexton, Mr. Wolfe and Mr. Gresham.

¹The following notations appear in this decision to identify exhibits: DX - Director exhibit; EX - Employer exhibit; CX - Claimant exhibit; and, TR - Transcript.

²I note that in *Braenovich v. Cannelton Industries, Inc.*, No. 02-0365 BLA (Feb. 12, 2003), the Benefits Review Board upheld an administrative law judge's equivalency finding that a 1.5 centimeter lesion on autopsy would constitute a lesion greater than one centimeter on a chest x-ray, even in the absence of a medical opinion on the radiographic equivalency. See also *Double B. Mining, Inc. v. Blankenship*, 177 F.3d 240, 244 (4th Cir. 1999) (two centimeters on autopsy was the minimum diameter for a lesion to be considered complicated pneumoconiosis) and *Clinchfield Coal Co. v. Fultz*, 61 Fed. Appx. 866, 871 (unpublished) (4th Cir. 2003) (medical evidence of the radiographic equivalency of a 1.2 to 1.3 centimeter autopsy mass is necessary; however, in other situations, some lesions may be "so large it is self-evident that they would have shown as opacities greater than one centimeter on x-ray. . .")

Evidentiary Discussion

At the hearing, I left the record open for Claimant to submit the deposition testimony of Dr. Perper, which Claimant had been unable to obtain prior to the hearing date. On July 28, 2004, I received the transcript from Dr. Perper's deposition taken on July 12, 2004. The exhibit is marked CX 2 and is admitted over Employer's objection to the submission of post hearing evidence. Accordingly, my decision in this case is based on the documents admitted into evidence (DX 1 to DX 76, CX 1, CX 2 and EX 1 to EX 5).

ISSUES

1. Whether the denial by the Benefits Review Board on October 30, 2002 of Mrs. Sexton's survivor claim should be modified due to a mistake of fact in its determination that Mr. Sexton's death was not due to coal workers' pneumoconiosis.

2. If Mrs. Sexton established a mistake of fact, whether she is entitled to survivor benefits under the Act.

FINDINGS OF FACT AND CONCLUSIONS OF LAW

Stipulations of Fact

At the hearing, the parties stipulated that: a) Mr. Sexton had at least 26 years of coal mine employment; b) Paramount Coal Co. is the responsible operator; and, c) Mrs. Sexton is an eligible survivor under the applicable regulations (TR, pages 8 to 12).

Coal Miner's Background

Born April 12, 1941, Mr. James Sexton married Mrs. Molly Sexton on July 14, 1984 (DX 2 and DX 7). From March 1973 until his death, Mr. Sexton worked for Paramount Coal Company, during which time he worked on the surface strip mines. His last job as a coal miner was as a front-end loader. During the last year and-a-half of his life, Mr. Sexton had breathing difficulties, which slowed down his active lifestyle. Mr. Sexton smoked an average of a pack and-a-half of cigarettes per day until 1980 (DX 47). On December 8, 1999, Mr. Sexton, struggling with shortness of breath was rushed to the hospital, and passed away on December 11, 1999 at the age of 58 (DX 8).

Issue # 1 – Modification of Survivor Claim Denial

To receive survivor benefits under the Act, and the implementing regulations, 20 C.F.R. § 718.205 (a), when a former coal miner's death was due to coal workers' pneumoconiosis, a surviving claimant must prove by a preponderance of the evidence several facts. First, the claimant must establish eligibility as a survivor. A surviving spouse may be considered eligible

for benefits under the Act if she was married to, and living with, the coal miner at the time of his death, and has not remarried.³

Next, the claimant must prove the coal miner had pneumoconiosis.⁴ “Pneumoconiosis” is defined as a chronic dust disease arising out of coal mine employment. The regulatory definitions include both clinical pneumoconiosis (the diseases recognized by the medical community as pneumoconiosis) and legal pneumoconiosis (defined by regulation as any chronic lung disease arising out of coal mine employment).⁵ The regulation further indicates that a lung disease arising out of coal mine employment includes “any chronic pulmonary disease or respiratory or pulmonary impairment significantly related to, or substantially aggravated by, dust exposure in coal mine employment.”⁶ As courts have noted, under the Act, the legal definition of pneumoconiosis is much broader than medical pneumoconiosis. *Kline v. Director, OWCP*, 877 F.2d 1175 (3d Cir. 1989).

Once a determination has been made that a miner had pneumoconiosis, it must be determined whether the coal miner's pneumoconiosis arose, at least in part, out of coal mine employment.⁷ If a miner who was suffering from pneumoconiosis was employed for ten years or more in one or more coal mines, there is a rebuttable presumption that pneumoconiosis arose out of such employment.⁸ Otherwise, the claimant must provide competent evidence to establish the relationship between pneumoconiosis and coal mine employment.⁹

Finally, the surviving spouse has to demonstrate the coal miner's death was due to pneumoconiosis.¹⁰

In summary, a survivor claim filed after January 1, 1982 must meet four primary elements for entitlement. The claimant bears the burden of establishing these elements by a preponderance of the evidence. If the claimant fails to prove any one of the requisite elements, the survivor claim for benefits must be denied. *Gee v. W. G. Moore and Sons*, 9 B.L.R. 1-4 (1986) and *Roberts v. Bethlehem Mines Corp.*, 8 B.L.R. 1-211 (1985). The first element relates

³20 C.F.R. § 718.4 indicates that the definitions in 20 C.F.R. § 725.101 are applicable. 20 C.F.R. § 725.101, in turn, refers to the term “survivor” as used in Subpart B of Part 725. 20 C.F.R. § 725.214 then sets out the spousal relationship requirements and 20 C.F.R. § 725.215 describes the dependency rules. According to § 725.214 (a) the spousal relationship exists if the relationship is a valid marriage under state law. Under § 725.215(a), a spouse is deemed dependent if she was residing with the miner at the time of his death.

⁴20 C.F.R. § 718.205 (a) (1) and see *Trumbo v. Reading Anthracite Co.*, 17 B.L.R. 1-85 (1993).

⁵20 C.F.R. § 718.201 (a) (1) and (2).

⁶20 C.F.R. § 718 (b).

⁷20 C.F.R. §§ 718.203 (a) and 205 (a) (2).

⁸20 C.F.R. § 718.203 (b).

⁹20 C.F.R. § 718.203 (c).

¹⁰20 C.F.R. § 718.205 (a) (3).

to the claimant's status as an eligible survivor of the deceased miner. The remaining three elements concern the deceased miner as follows: the coal miner suffered from pneumoconiosis; the coal miner's pneumoconiosis arose out of coal mine employment; and, the coal miner's death was due to coal workers' pneumoconiosis.

In his adjudication of Mrs. Sexton's survivor claim, Judge Miller determined Mr. Sexton's death was due to coal workers' pneumoconiosis because the evidence established the presence of complicated pneumoconiosis in his lungs which in turn invoked an irrebuttable regulatory presumption of death due to pneumoconiosis. However, the Benefits Review Board disagreed and concluded the medical evidence did not establish the presence of complicated pneumoconiosis.¹¹ As a result, Mrs. Sexton was unable to invoke the complicated pneumoconiosis regulatory causation presumption and correspondingly failed to establish that her husband's death was due to coal workers' pneumoconiosis. Accordingly, the Board denied her survivor claim. Subsequently, Mrs. Sexton presented additional medical evidence in an effort to modify the adverse decision on the basis of mistake of fact.

Any party to a proceeding may request modification at any time before one year from the date of the last payment of benefits or at any time before one year after the denial of a claim. 20 C.F.R. § 725.310 (a).¹² Upon the showing of a "change in conditions" or a "mistake in a determination of fact," the terms of an award or the decision to deny benefits may be reconsidered. 20 C.F.R. § 725.310. An order issued at the conclusion of a modification proceeding may terminate, continue, reinstate, increase or decrease benefit payments or award benefits. Since a survivor claim involves a deceased miner, the change in conditions provision is not applicable. Thus, the sole inquiry is whether a mistake of fact of was made during the final prior adjudication.

In considering whether a mistake of fact was made during the Board's denial of Mrs. Sexton's survivor claim, I will consider the entire evidentiary record and the newly submitted evidence,¹³ which includes additional medical reviews of evidence that was in existence during the previous adjudication of this claim, to determine whether Mr. Sexton's death was due to pneumoconiosis.

¹¹The Board also affirmed Judge Miller's determination under 20 C.F.R. §§ 718.205 (c) (1) and (2) that coal workers' pneumoconiosis neither caused nor was a significant contributing factor in Mr. Sexton's death (DX 69).

¹²In January 2001, a new set of DOL regulations concerning the adjudication of black lung claims became effective. Only some portions of the new regulations are applicable to Mrs. Sexton's modification request of her survivor claim since it relates to a claim that was not finally denied more than one year prior to January 2001; such applicable provisions will be designated with "(2001)" as a suffix. (*see* 20 C.F.R. § 725.2 (c) (2001)).

¹³*See Kingery v. Hunt Branch Coal Co.*, 19 B.L.R. 1-6 (1994).

Death Due to Pneumoconiosis

For a survivor claim filed on or after January 1, 1982, the Department of Labor regulations provide four means to establish that a coal miner's death was due to coal workers' pneumoconiosis:¹⁴

1. The miner had complicated pneumoconiosis;¹⁵
2. Death was caused by pneumoconiosis;
3. Death was caused by complications of pneumoconiosis; or,
4. Pneumoconiosis was a substantially contributing cause or factor leading to the miner's death. Notably, pneumoconiosis is deemed to be a substantially contributing cause of a miner's death if it hastens the miner's death.¹⁶

However, a survivor may not receive benefits if the coal miner's death was caused by traumatic injury, or the principal cause of death was a medical condition not related to pneumoconiosis, unless evidence establishes that pneumoconiosis was a substantially contributing cause of death.

Complicated Pneumoconiosis

The regulation, in part, at 20 C.F.R. § 718.304, provides that if a claimant is able to establish the presence of complicated pneumoconiosis, then an irrebuttable presumption of death due to pneumoconiosis is established. In the Black Lung Benefits Act, 30 U.S.C. 921 (c) (3) (A) and (C), as implemented by 20 C.F.R. § 718.304 (a), Congress determined that if a miner suffered from a chronic dust disease of the lung which "when diagnosed by chest X-ray, yields one or more large opacities (greater than one centimeter in diameter) and would be classified in category A, B, or C," there shall be an irrebuttable presumption that his death was due to pneumoconiosis.¹⁷ This type of large opacity is called "complicated pneumoconiosis." 20

¹⁴20 C.F.R. §§ 718.205 (c) (1), (2), and (3), and 304.

¹⁵According to 20 C.F.R. § 718.304, if a miner had complicated pneumoconiosis, an irrebuttable presumption exists that his death was due to pneumoconiosis.

¹⁶20 C.F.R. § 718.205 (c) (5). Prior to publication of the new regulations, the U.S. Court of Appeals for the Fourth Circuit, like other federal circuits, interpreted "substantially contributing cause" to include a hastening of a miner's death. *Shuff v. Cedar Coal Co.*, 967 F.2d 977, 980 (4th Cir. 1992). Under this interpretation, any acceleration of the miner's death that is attributable to pneumoconiosis will entitle a claimant to survivor benefits.

¹⁷On the standard ILO chest x-ray classification worksheet, Form CM 933, large opacities are characterized by three sizes of opacities, identified by letters. The interpretation finding of Category A indicates the presence of a large opacity having a diameter greater than 10 mm (one centimeter) but not more than 50 mm; or several large opacities, each greater than 10 mm but the diameter of the aggregate does not exceed 50 mm. Category B mean an opacity, or opacities "larger or more numerous than Category A" whose combined area does not exceed the equivalent of the right upper zone of the lung. Category C represents one or more large opacities whose combined area exceeds the equivalent of the right upper zone.

C.F.R. §§ 718.304 (b) and (c) also permits complicated pneumoconiosis to be established by either the presence of massive fibrosis in biopsy and autopsy evidence or other means which would be expected to produce equivalent results in chest x-rays or biopsy/autopsy evidence.

According to the U.S. Court of Appeals for the Fourth Circuit¹⁸ in *Eastern Associated Coal Corp. v. Director, OWCP [Scarbro]*, 220 F.3d 250 (4th Cir. 2000), the existence of complicated pneumoconiosis is established by “congressionally defined criteria.” As a result, the statute’s definition of complicated pneumoconiosis as radiographic evidence of one or more large opacities categorized as size A, B, or C, 30 U.S.C. 921 (c) (3) (A), represents the most objective measure of the condition. This sets the benchmark by which other methods for proving complicated pneumoconiosis are measured, as described in 30 U.S.C. 921 (c) (3) (B) and (C). *Id.* at 256. In other words, whether a massive lesion or other diagnostic results represent complicated pneumoconiosis under 30 U.S.C. 921 (c) (3) (B) and (C) requires an equivalency evaluation with the x-ray criteria set forth in 30 U.S.C. 921 (c) (3) (A).¹⁹ Additionally, the court emphasized that the legal definition of complicated pneumoconiosis as established by Congress controls over the medical community’s definition of the disease. *Id.* at 257. Finally, the court indicated that although all relevant and conflicting medical evidence must be considered and evaluated:

if the x-ray evidence vividly displays opacities exceeding one centimeter, its probative force is not reduced because the evidence under some other prong is inconclusive or less vivid. Instead, the x-ray evidence can lose force only if other evidence affirmatively shows that the opacities are not there or are not what they seem to be, perhaps because of an intervening pathology, some technical problem with equipment, or incompetence. *Id.*

In light of these statutory, regulatory and judicial principles, the adjudication of whether a claimant is able to invoke the irrebuttable presumption under 20 C.F.R. § 718.304 involves a two-step process. First, I must determine whether: a) the preponderance of the chest x-rays establishes the presence of large opacities characterized by size as Category A, B, or C under recognized standards; or b) biopsy or autopsy evidence or other diagnostic means discloses massive lesions which are equivalent to chest x-ray evidence of large opacities characterized as Category A, B, or C. At this stage of the process, the essential inquiry is whether such large opacities, or their equivalent, exist. Thus, as observed by the *Scarbro* court, definitive evidence indicating the large opacities are not really present would preclude invocation of the 20 C.F.R. § 718.304 presumption.

Second, if the preponderance of the evidence demonstrates the existence of large opacities, I must then consider all other relevant evidence to determine whether that evidence affirmatively shows the large opacities are not what they seem to be due to some other pathology.

¹⁸Mrs. Sexton’s case arises within the jurisdiction of this court.

¹⁹See also 20 C.F.R. §§ 718.304 (b) and (c).

Existence of Large Opacities

Chest X-Rays

First, I will evaluate chest x-ray imaging under 20 C.F.R. § 718.304 (a) to determine whether large opacities are present. The radiographic evidence in the record is set out below.

Date of x-ray	Exhibit	Physician	Interpretation
June 8-23, 1980 (multiple x-rays)	DX 38, 42	multiple physicians	(Negative) pneumothorax present to clearing, mediastinal emphysema and fibrosis present and infiltrate in left lower lung field
August 23, 1985	DX 38	Dr. DePonte, BCR, B ²⁰	(Negative) bullous and fibrotic changes of left lung consistent with known mycobacterium infection, some emphysema with hyperinflation and attenuation of interstitial markings
August 4, 1986	DX 38	Dr. DePonte, BCR, B	(Negative) several bullae, changes of chronic COPD (chronic obstructive pulmonary disease) present, no acute abnormality
December 2, 1987	DX 38	Dr. DePonte, BCR, B	(Negative) bullous and fibrotic changes of left lung consistent with known mycobacterium infection, scarring in left lung and underlying changes of COPD
September 24, 1999	DX 43	Dr. Scott, BCR, B	Negative for pneumoconiosis, scarring and infiltrates with pleural thickening compatible with tuberculosis, unknown activity
(same)	DX 14	Dr. DePonte, BCR, B	(Negative) progressive scarring does not indicate active disease; underlying changes of obstructive airways disease present
(same)	DX 32	Dr. Spitz, BCR, B	Negative for pneumoconiosis, right upper lobe volume loss with infiltrate and minimal pleural thickening consistent with active tuberculosis
(same)	DX 32	Dr. Wiot, BCR, B	Negative for pneumoconiosis, inflammatory process with most likely active tuberculosis process, changes on right consistent with old granulomatous disease and bullae formation, pleural thickening
November 26, 1999	DX 43	Dr. Wheeler, BCR, B	Negative for pneumoconiosis, moderate interstitial infiltrates, “check for active tuberculosis”
(same)	DX 15	Dr. DePonte, BCR, B	(Negative) progressive left lung pneumonia superimposed on underlying chronic changes with pleural thickening, no evidence of congestive failure
(same)	DX 43	Dr. Scott, BCR, B	Negative for pneumoconiosis, new infiltrates, probably pneumonia or tuberculosis, fibrosis and pleural thickening probably due to tuberculosis, unknown activity

²⁰The following designations apply: B – B reader, and BCR – Board Certified Radiologist. These designations indicate qualifications a person may possess to interpret x-ray film. A “B Reader” has demonstrated proficiency in assessing and classifying chest x-ray evidence for pneumoconiosis by successful completion of an examination. A “Board Certified Radiologist” has been certified, after four years of study and examination, as proficient in interpreting x-ray films of all kinds including images of the lungs. *See also* 20 C.F.R. § 718.202 (a) (1) (ii).

(same)	DX 32	Dr. Spitz, BCR, B	Negative for pneumoconiosis, right upper lobe volume loss with infiltrate and minimal pleural thickening; progression of disease on lung consistent with active tuberculosis
November 29, 1999	DX 43	Dr. Scott, BCR, B	Negative for pneumoconiosis, infiltrates and/or fibrosis, bullous emphysema
(same)	DX 16	Dr. DePonte, BCR, B	(Negative) stable bilateral pulmonary infiltrates related to bacterial process or known atypical mycobacterium infection
(same)	DX 32	Dr. Spitz, BCR, B	Negative for pneumoconiosis, right upper lobe volume loss with infiltrate and minimal pleural thickening; progression of disease on lung consistent with active tuberculosis
(same)	DX 32	Dr. Wiot, BCR, B	Negative for pneumoconiosis, inflammatory process with most likely active tuberculosis process, bullae present
December 3, 1999	DX 43	Dr. Scott, BCR, B	Negative for pneumoconiosis, infiltrates and/or fibrosis, bullous emphysema present and pleural thickening and volume compatible with tuberculosis, unknown activity
(same)	DX 43	Dr. Wheeler, BCR, B	Negative for pneumoconiosis, ill defined interstitial infiltrates
(same)	DX 17	Dr. DePonte, BCR, B	(Negative) interval part clearing of left lung pneumonia, no new or progressive infiltrates
(same)	DX 18, 21	Dr. Peter Barrett, BCR, B	Negative for pneumoconiosis, emphysema, bullae and probably old tuberculosis present
(same)	DX 32	Dr. Spitz, BCR, B	Negative for pneumoconiosis, progression of disease into right lower lung consistent with active tuberculosis, development of additional pleural thickening
(same)	DX 32	Dr. Wiot, BCR, B	Negative for pneumoconiosis, inflammatory process with most likely active tuberculosis process, bullae present
December 8, 1999	DX 44	Dr. Wiot, BCR, B	Negative for pneumoconiosis
(same)	DX 20	Dr. Barrett, BCR, B	Negative for pneumoconiosis, bullae, emphysema and tuberculosis present, also right lower lung pneumonia
(same)	DX 13, 19	Dr. DePonte, BCR, B	(Negative) interval development of right lower lobe pneumonia with mixed interstitial and alveolar infiltrate
(same)	DX 43	Dr. Wheeler, BCR, B	Negative for pneumoconiosis, moderate interstitial infiltrates compatible with pneumonia
(same)	DX 43	Dr. Scott, BCR, B	Negative for pneumoconiosis, new infiltrate probably due to pneumonia, pleural thickening and volume compatible with tuberculosis, unknown activity and bullous emphysema present
(same)	DX 32	Dr. Spitz, BCR, B	Negative for pneumoconiosis, further progression of disease in right lower lung consistent with active tuberculosis
(same)	DX 34	Dr. Wiot, BCR, B	Negative for pneumoconiosis, bullae present, diffuse infection now bilateral
December 11, 1999	DX 13, 22	Dr. DePonte, BCR, B	(Negative) left mid and upper lung zones show changes of diffuse pleural thickening with bullae

None of the ten chest x-rays in the record is positive for the presence of a large opacity. Therefore, the preponderance of the radiographic evidence does not establish the presence of a large opacity in Mr. Sexton's lungs. However, Mrs. Sexton may still establish the presence of a large opacity in her husband's lungs through autopsy or other means.

Autopsy Evidence

Evaluation of the autopsy evidence has two parts. First, I must determine whether a large pulmonary nodule or mass was identified during the autopsy. Second, if such a mass was found, the evidentiary record must also support a finding that the mass would appear on a chest x-ray as an opacity greater than one centimeter.

1. Large Pulmonary Nodule

(Note: the following medical summaries, and other remaining portions of this decision, contain detailed information obtained from the autopsy of Mr. Sexton, submitted by Mrs. Sexton to support her survivor claim. While respecting the dignity and privacy of the deceased, some discussion of the detailed observations is necessary because I find the medical information relevant on determining whether Mr. Sexton's death was due to pneumoconiosis.)

Prior to reviewing the diverse reports concerning Mr. Sexton's autopsy and the associated examination of his lung tissue, a review of the regulatory provisions on the requisite standard for diagnosing pneumoconiosis based on autopsy or biopsy helps to understand the significance of some of the reports. The regulations define "clinical" pneumoconiosis as a condition characterized by permanent deposition of substantial amounts of particulate matter, caused by coal dust exposure, in the lungs and "the fibrotic reaction of the lung tissue to that deposition." 20 C.F.R. § 718.201 (a) (emphasis added). As a result, an autopsy or biopsy finding of anthracotic pigmentation, standing alone, is not sufficient to establish the presence of pneumoconiosis, 20 C.F.R. § 718.202 (a) (2). Additionally, a diagnosis of a "chronic lung disease" (complicated pneumoconiosis) may be established if a biopsy or autopsy reveals the presence of massive lesions. 20 C.F.R. § 718.304.

Dr. Joan C. Coogan
(DX 9 and DX 10)

On December 12, 1999, Dr. Coogan conducted an autopsy of Mr. Sexton, consisting of gross and microscopic examinations.

Upon gross examination of the heart, Dr. Coogan discovered that the aorta contained a moderate amount of atherosclerotic plaque formation. The iliac arteries also showed bilateral and "very prominent" plaque formation.

The apex of the right lung was scarred and contained a cyst formation. Dr. Coogan noted, "around this area, there is prominent paraseptal anthracosis with macule formation. These macules coalesce, occasionally reaching a diameter of 2 cm." The left lung displayed severe dense adhesions, apical scarring with cyst and "prominent macule formation." Additionally, Dr.

Coogan found severe bronchopneumonia, with the left bronchial tree full of “purulent” material. Finally, multiple pulmonary lymph nodes were anthracotic.

The gross evaluation of the right hemisphere of the brain suggested the presence of an “acute non-hemorrhagic infarct, secondary to a vascular thrombosis.” Specifically, Dr. Coogan observed the right hemisphere was “diffusely swollen” and “markedly congested,” with the inferior cerebellum depressed bilaterally due to a herniation. The right internal carotid artery was “completely occluded by a pale staining clot.”

Under the microscope, in the heart tissue, Dr. Coogan confirmed the presence of plaque material and “recent clot formation.”

Microscope slides of the right lung samples contained evidence of diffuse pulmonary congestion. Dr. Coogan also saw “multiple, variably-sized densely hyalinized macules with coalesce . . . associated with anthrasilicotic pigment deposition,” consistent with coal workers’ pneumoconiosis. The left lung tissue also contained multiple anthrasilicotic macules and multiple areas of bronchopneumonia. The apex cystic activity associated with the pulmonary scarring in the left lung revealed nodules consistent with aspergillosis.²¹

The right hemisphere carotid artery tissue showed evidence of an “ante-mortem” event. The left brain tissue also demonstrated “extensive ischemic damage consistent with acute injury.”

Based on her evaluation, Dr. Coogan concluded that Mr. Sexton died due to a stroke. At the time of his death, Mr. Sexton also had pneumonia and black lung disease. Dr. Coogan’s final pathological diagnosis included: 1) extensive right cerebral ischemic changes compatible with an acute non-hemorrhagic cerebral vascular accident secondary to right internal carotid artery thrombosis; 2) focal early ischemic changes involving the left cerebral hemisphere, cerebellum and upper cervical spinal cord; 3) left lung bronchopneumonia; 4) extensive alveolar damage with hyaline membranes, right lung; 5) bilateral anthrasilicosis compatible with coal workers’ pneumoconiosis; 6) severe left pulmonary pleura adhesions; 7) aspergilloma of left upper lung arising in cavitary pulmonary scarring; 8) chronic passive congestion; and, 9) localized severe atherosclerosis involving aorta and bilateral iliac arteries.

Dr. Richard Naeye
(DX 11, DX 42, and EX 5)

On May 2, 2000, Dr. Richard Naeye, board certified in anatomical and clinical pathology, reviewed the final hospital records and examined seven lung slides. According to Dr. Naeye, Mr. Sexton, who worked in a coal strip mine and was a non-smoker, entered the hospital on December 8, 1999 with pneumonia. Although the pneumonia appeared to respond to antibiotics after two days, Mr. Sexton developed a thrombosis in the right internal carotid artery, which caused severe mental impairment and confusion and led to cardiac arrhythmia and death.

²¹A disease condition caused by a fungus, *Aspergillus*, “marked by inflammatory granulomatous lesions in the . . . lungs.” DORLAND’S ILLUSTRATED MEDICAL DICTIONARY 148 (28th ed. 1994).

Through the microscope, Dr. Naeye observed three pulmonary abnormalities. First, he found old masses of hyalinized collagen with admixed black pigment and birefringent crystals of all sizes, several of which exceeded one centimeter and may have reached two centimeters. Second, a cavitary lesion caused by fungus infection (aspergillus) was present. And, third, he found very recent onset of acute lobular pneumonia, characterized by foci of acute inflammatory cells filling alveolar lumina with adjacent areas of edema in multiple lobes of the lungs. While some chronic bronchitis was present, Dr. Naeye did not see chronic emphysema. Although the silicotic lesions were not sufficient for a medical diagnosis of complicated coal workers' pneumoconiosis, Dr. Naeye opined that since the lesions exceeded one centimeter and may have reached two centimeters, they met the current legal definition of complicated pneumoconiosis.

At a deposition taken on September 29, 2000, having reviewed Dr. Coogan's autopsy report, Dr. Naeye provided additional information concerning the three pulmonary abnormalities that he observed during his pathologic study of the lung tissue. First, the "hyalinized masses of collagen" contained many birefringent crystals, which were at least partially silicotic in origin. While several lesions exceeded one centimeter in diameter, he was uncertain whether any reached two centimeters in diameter because the slides were not large enough to accommodate lesions that large. At the same time, Dr. Naeye did not challenge Dr. Coogan's finding of two centimeter silicotic lesions on gross examination.

The second pulmonary abnormality might have appeared to be complicated pneumoconiosis on gross examination. However, a microscopic evaluation revealed the large cavitary lesion present at autopsy was due to a fungal infection, specifically aspergillus. The fungus is unrelated to coal mine employment.

The third pulmonary finding was rapidly spreading acute lobular pneumonia, which represented a terminal event. Dr. Naeye did not believe Mr. Sexton's pneumonia was related to his employment as a miner.

In medical terms, "complicated pneumoconiosis is an immunologic disorder that can arise out of really mild simple coal workers' pneumoconiosis." Based on his decades of experience and as a pathologist, Dr. Naeye opined that "none of the lesions" in Mr. Sexton's case were "characteristic of complicated coal workers' pneumoconiosis." Notably, complicated pneumoconiosis destroys blood vessels during its inflammatory stage which causes a "classic" complicated pneumoconiosis lesion to "fill with black liquid." In Mr. Sexton's case, the lesions did not contain such fluid. Instead, the noted lesions were consistent with "free silica and some of the free radicals that are released from coal when its first taken out of the seam." Consequently, Dr. Naeye would not render a medical diagnosis of complicated pneumoconiosis. Dr. Naeye agreed that Mr. Sexton had simple coal workers' pneumoconiosis. Based on what lawyers have told him, Dr. Naeye diagnosed legal complicated pneumoconiosis because the lesions were greater than one centimeter.

Dr. Naeye stated that the microscopic lesions "probably" would not show up as an opacity greater than one centimeter on a chest x-ray. He was uncertain since no scientific study had evaluated the pathologic and related radiographic sizes of black lung lesions. While acknowledging that pneumoconiosis may be present even in the absence of radiographic

evidence, Dr. Naeye nevertheless noted that Dr. Wiot did not observe any opacities greater than one centimeter in Mr. Sexton's chest x-rays.

Reiterating that pneumonia was the terminal event for Mr. Sexton, Dr. Naeye opined that the simple coal workers' pneumoconiosis did not contribute to the miner's death because Mr. Sexton did not have any significant respiratory disability. Mr. Sexton worked until he was hospitalized and produced near normal results on a lung function test completed five weeks before he died. Dr. Naeye also noted the normal findings on chest x-ray reports.

On cross-examination, after agreeing that Mr. Sexton had simple coal workers' pneumoconiosis even though the chest x-rays were negative, Dr. Naeye stated that microscopic evaluation was more specific than chest x-rays for a diagnosis. The microscopically observed crystal mixed with black pigment represented simple coal workers' pneumoconiosis.

Due to the limits of the cover slip on a microscopic slide, Dr. Naeye could not measure the lesions beyond 1.2 centimeters. At the same time, if it appears that half the lesion is on the slide, a physician might postulate as to the size of the whole lesion. Additionally, Dr. Naeye did not doubt Dr. Coogan's gross examination finding of lesions up to two centimeters.

Dr. Naeye disagreed that a two centimeter lesion found during an autopsy represented complicated pneumoconiosis. He explained that, although the lesions in Mr. Sexton's case may have reached two centimeters, the presence of these lesions did not warrant a diagnosis of complicated pneumoconiosis, which is a different disease process from simple coal workers' pneumoconiosis. Complicated pneumoconiosis has a different genesis. In simple coal workers' pneumoconiosis, the micronodules or macronodules expand slowly over time and come together to form a confluent mass. Even if these confluent masses reach two centimeters, that is not sufficient to diagnose complicated pneumoconiosis, which is a different disease with a different cause. Complicated pneumoconiosis is a disease that continuously expands and is an immunological disorder, while simple coal workers' pneumoconiosis will stop progressing the day a miner ceases coal mine employment. The lesions of simple coal workers' pneumoconiosis, therefore, do not expand after cessation of exposure to coal mine dust. Dr. Naeye also agreed it is not common, but it is possible, for a miner to have normal lung function even with complicated pneumoconiosis, and he agreed it is well recognized that many coal workers' pneumoconiosis lesions can not be seen on chest x-ray readings.

While agreeing that chest x-rays may understate the size of a lesion, Dr. Naeye reiterated that no one could state with certainty that a two centimeter lesion would show up as a one centimeter opacity on a x-ray film. Based on the negative readings he reviewed, which showed no evidence of coal workers' pneumoconiosis, he opined that the perfusion of pulmonary lesions was low. If the number of lesions present were of a greater perfusion, the pneumoconiotic lesions would appear on a chest x-ray. Finally, Dr. Naeye concurred with Dr. Coogan's conclusion that although Mr. Sexton had pneumoconiosis, a stroke caused his death.

Dr. Naeye reviewed additional medical records, which included Dr. Perper's and Dr. Bush's medical reports, and testified in another deposition conducted on May 25, 2004. Dr. Naeye continued to believe that the microscopic nodules he evaluated would not appear as one

centimeter opacities on a chest x-ray. About a month prior to Mr. Sexton's death, pulmonary function tests demonstrated that he was not totally disabled. Mr. Sexton was hospitalized in December 1999 after he became short of breath due to pneumonia. The abnormal blood gas studies at that time were due to his pneumonia.

Dr. Naeye had previously indicated the "near absence" of emphysema in Mr. Sexton. The physician explained that while the lung tissue samples contained evidence of "severe" emphysema, Dr. Naeye was referring to clinical, rather than anatomical, emphysema. That is, since Mr. Sexton did not have a disabling pulmonary impairment, his clinical emphysema was not significant.

Dr. Naeye pointed out that while Dr. Perper emphasized the lack of arterial sclerosis in the carotid arteries, Mr. Sexton's thrombus could have been associated with arterial sclerosis in some other artery. Coal workers' pneumoconiosis does not cause thrombosis. Mr. Sexton's thrombus and associated cerebral vascular accident and pneumonia were individually "highly significant and very dangerous disorders."

Dr. Naeye reviewed the microphotographs that Dr. Perper made from the lung slides and opined that there were no lesions of complicated coal workers' pneumoconiosis present. Mr. Sexton's pleura was thickened but Dr. Naeye attributed that to Mr. Sexton's old infection, which was thought to be tuberculosis originally or the abrasive substance put on his pleura to avoid a recurrence of the pneumothorax.

On cross-examination, Dr. Naeye believed a single autopsy nodule measured as two centimeters would appear to be at least one centimeter on a x-ray. At the same time, Dr. Naeye noted that an agreement had developed in the medical community that a two centimeter nodule discovered during an autopsy would be "not much more than one centimeter" on a chest x-ray.

The lesions in Mr. Sexton's chest were multiple old nodules that slowly grew together. In contrast, complicated pneumoconiosis lesions grow rapidly over the course of several years. Mr. Sexton's lesions were more likely due to bacterial infection. At the same time, the lesions also contained silica crystals indicating the lesions had "more than one origin." Dr. Naeye acknowledged that complicated pneumoconiosis lesions frequently develop in the upper lung lobes. Consequently, the location of Mr. Sexton's pulmonary lesions is consistent with complicated pneumoconiosis. The physician also agreed that many people with complicated pneumoconiosis do not show a pulmonary impairment. Mr. Sexton did not have a significant enough pulmonary disability to prevent him from doing hard physical work. Generally, miners with complicated pneumoconiosis are more susceptible to pneumonia; however, in Mr. Sexton's case, he had normal pulmonary function, so his defense mechanisms were not compromised. Where nodules were present in the lung is a non-functional part of the lung because the lung tissue has been destroyed. Dr. Naeye does not believe the nodules prevented Mr. Sexton from recovering from the pneumonia because his condition deteriorated so rapidly. Dr. Naeye stated that he is not sure whether the confluence of nodules reached two centimeters because there was not enough tissue on the slides to make that determination. Finally, after indicating that he was aware of Dr. Wiot's negative chest x-rays findings, Dr. Naeye also indicated that he would have expected the significant silica lesions he observed on the lung tissue slides to have shown up on a

chest x-ray. Regardless, of the chest x-rays evidence, Dr. Naeye emphasized that Mr. Sexton had good lung function until an “acute illness” killed him.

Steven T. Bush
(EX 1 and EX 2)

On April 22, 2004, Dr. Bush, board certified in pathology, reviewed Mr. Sexton’s medical records and evaluated the lung slides. Dr. Bush reported that Mr. Sexton had a history of a pneumothorax following a minimum trauma at work in 1980 and pulmonary tuberculosis in 1984. He smoked a pack of cigarettes a day for fifteen years. During Mr. Sexton’s final hospital admission, he was treated for pneumonia but suffered a catastrophic stroke from thrombosis of the right carotid artery.

A review of all the autopsy slides and medical records show that Mr. Sexton had coal workers’ pneumoconiosis manifested by black dust pigment of mild to moderate degree associated with dense fibrous scarring with scar emphysema. A moderately large number of silica and silicate particles were present. The lesions of pneumoconiosis were focused in the right and left apices. Dr. Bush noted that progressive massive fibrosis lesions are “almost invariably heavily pigmented” but the lesions in Mr. Sexton’s lungs were only mildly pigmented and not massive. The lesions were pleural based and, though somewhat elongated, no more than 0.9 centimeters wide. According to Dr. Bush, the lesions are not coalescent but are individual lesions near, and sometimes adjacent to, one another. Dr. Bush attributed the pleural fibrosis in the left lung to the 1980 surgery to prevent repeated pneumothorax.

Dr. Bush opined that the extent of pneumoconiosis was too limited to have produced radiological changes of pneumoconiosis or physiologic respiratory impairment. The respiratory symptoms suffered by Mr. Sexton during his final hospital admission were caused by bronchopneumonia (the left lung showed “severe, acute bronchopneumonia”) and his death was eventually caused by cerebral edema from the stroke. A colonizing fungus ball (Aspergilloma) was also present. Dr. Bush estimated that pneumoconiosis affected only 5 to 10 percent of Mr. Sexton’s lungs. The physicians attributed Mr. Sexton’s death to a massive stroke, which occurred during Mr. Sexton’s hospitalization for treatment of bronchopneumonia. The factors causing the stroke are not known. Based on the chest x-rays, gross autopsy findings, and his examination of the lung tissue slides, Dr. Bush opined the pneumoconiosis was too limited to be related to the stroke or the bronchopneumonia. Dr. Bush concluded that Mr. Sexton would have died of a massive stroke had he never been exposed to the pulmonary hazards associated with coal mine employment.

On May 10, 2004, Dr. Bush reviewed Dr. Perper’s medical report dated April 14, 2004. Dr. Bush disagreed with his diagnosis of progressive massive fibrosis. The description of Mr. Sexton’s lungs on autopsy describes changes “essentially confined to the apex” and the gross description of an area two centimeters in size showing apparent coalescence is not borne out on the histologic slides. The slides show clusters of lesions and scarring in the apex. The history of pleural abrasions to induce scarring of the pleura would cause some of these changes observed on autopsy and would arise from pneumothorax incidences.

Dr. Bush noted that the cluster of lesions in the apex, some consistent with coal workers' pneumoconiosis and some fibrotic areas consistent with silicosis and others that are scarring are too limited in extent or size to merit a diagnosis of progressive massive fibrosis. The lesions in Mr. Sexton's chest were first noted in 1980 and did not significantly advance in 20 years. Consequently, the lesions were not progressing; instead, they were repeatedly deemed "stable." Dr. Bush pointed out that Dr. Perper did not consider Mr. Sexton's "heavy smoking history." Dr. Bush believed Dr. Perper incorrectly assessed the two centimeter length of the lesions because the lesions appeared elongated from the magnification, rather than acutely displaying their true dimensions.

Dr. Bush recognized that the lesions demonstrate the presence of coal workers' pneumoconiosis but because the gross pathologic changes outside of the apices, throughout the rest of the lungs had only mild dust pigmentation and hemorrhagic discoloration of the cut surface was red-yellow, there was no evidence of massive pulmonary fibrosis. In contrast, progressive massive fibrosis has heavy pigmentation and associated appearance of black on surface and cut sections.

Dr. Bush disagrees with Dr. Perper attributing Mr. Sexton's shortness of breath during his last hospital admission as evidence of pneumoconiosis. In Dr. Bush's opinion, the lung symptoms developed from acute bronchopneumonia. Based on several medical studies and Mr. Sexton's hypoxemia, Dr. Perper links Mr. Sexton's cardiac arrhythmia suffered during the last moments of his life to chronic obstructive pulmonary disease. However, Dr. Bush observes that Dr. Perper has focused on only one of many factors that could have played a role in Mr. Sexton's death. Because there are numerous possible causes for the cardiac arrhythmia, Dr. Perper's conclusion would only be valid if there was extensive disease in Mr. Sexton's lungs and lifetime pulmonary evaluations showing significant impairment of pulmonary function. Yet, considering the chest x-rays, clinical findings, and autopsy results, Dr. Bush believes his assessment of lung damage in the range of 5 to 10% is more accurate. Thus, Mr. Sexton was not disabled from a pulmonary standpoint and even worked until his final hospital admission. In Dr. Bush's opinion, Dr. Perper overstates the severity of Mr. Sexton's pneumoconiosis, incorrectly links his obstructive pulmonary disorder to the cerebral stroke, and inappropriately ties the cardiac abnormalities to the chronic lung disease.

Finally, Dr. Bush does not believe that the moderate degree of pneumoconiosis evident in the lungs caused, contributed to, or hastened Mr. Sexton's death. Mr. Sexton developed bronchopneumonia, which is not restricted to individuals with occupational dust exposure, suffered a massive stroke, and died when efforts to assist his respiration failed.

Dr. Joshua A. Perper
(CX 1 and CX 2)

In April 2004, Dr. Perper, board certified in anatomical, surgical and forensic pathology, reviewed the following records and materials: copy of Mr. Sexton's death certificate, autopsy by Dr. Coogan, Dr. Naeye's evaluation of the autopsy evidence, microscopic slides of the autopsy, reports of chest x-ray interpretations by multiple physicians, and treatment/hospital records from 1980 to 1999, which contained pulmonary function tests. Mr. Sexton had mined

coal above ground for 26 to 34 years. He had smoked cigarettes at the rate of one to one and a half packs a day for 15 to 20 years. His medical history was significant for a chest injury and collapsed left lung in 1980, a diagnosis of possible tuberculosis in 1984, recurrent pneumonia, and minimal obstructive pulmonary disease. In December 1999, Mr. Sexton presented at the emergency room with sudden onset of increased shortness of breath. While being treated for pneumonia and chronic obstructive pulmonary disease, Mr. Sexton suffered a stroke, required respiration assistance, developed cardiac arrhythmia, and died.

Under the microscope, Dr. Perper observed marked thickening and fibrosis of pleura with fibro-anthracosis and pleural/subpleural silicotic type of nodule extending into the parenchymal lung tissue. Dark anthracotic pigmentation is observed. Beneath the pleural, there is evidence of scar (focal) emphysema and centrilobular emphysema. Dr. Perper stated, "the pneumoconiotic nodules and areas of fibro-anthracosis measure up to more than 2.0 cm, and/or replace more than 80-90% of the lung section. In places it is clear that the pneumoconiotic lesion extends beyond the edges of the lung section." Another tissue slide shows hyaline-anthracotic areas with perivascular chronic infiltrates and a perivascular anthracotic macule. In another slide, an anthracotic macule around a blood vessel shows severe sclerosis and narrowing of lumen. Beneath the macule there is a mixed coal dust type of micronodule and above it is interstitial fibro-anthracosis. At a higher magnification, Dr. Perper observed an anthraco-silicotic nodule showing anthracotic pigmentation in the hyaline core and the peripheral fibro-anthracotic layers. He additionally observed acute bronchopneumonia and recent thrombus in the carotid artery section which also had normal walls and no arteriosclerosis.

Based on his microscopic evaluation, Dr. Perper diagnosed severe complicated pneumoconiosis "on the background of simple pneumoconiosis." Mr. Sexton also had acute bronchopneumonia, slight to moderate centrilobular emphysema, and "ischemic changes of the brain."

Dr. Perper concluded that Mr. Sexton acquired coal workers' pneumoconiosis, having worked for more than 26 years and perhaps as many as 34 years in coal mining. His worsening chronic obstructive lung disease with marked shortness of breath, decreased breath sounds, coughing, and chronic bronchitis indicated the presence of pneumoconiosis. Also, he had abnormal pulmonary function studies and hypoxemia in the month preceding death. His hypoxemia ultimately required administration of bronchodilators, steroids and continuous supplemental oxygen. Some radiologists read chest x-rays as negative for pneumoconiosis but found evidence of granulomatous disease or tuberculosis; however, no granulomas or observations indicative of tuberculosis were found when the autopsy was conducted. Massive pneumoconiotic lesions were found on autopsy, leading to the "inescapable conclusion that the process was clearly and unequivocally complicated coal workers' pneumoconiosis." Dr. Perper believed the radiological severity and magnitude of opacities, relentless progression and deterioration of pulmonary function in a relatively young miner who stopped smoking more than a decade before his death were consistent with a finding of complicated pneumoconiosis. Dr. Perper discounts the re-readings of the chest x-rays by Dr. Spitz and Dr. Wiot after Mr. Sexton's death because the physicians found the presence of tuberculosis, which was not observed during the autopsy.

Moreover, in addition to macular and micronodular lesions consistent with moderate coal workers' pneumoconiosis, the autopsy prosector observed large macronodular fibro-anthracotic lesions of anthraco-silicosis, exceeding two centimeters, consistent with complicated coal workers' pneumoconiosis. Based on medical literature, Dr. Perper believes progressive massive fibrosis is by definition present when a lesion that is at least two centimeters in diameter is present.

Dr. Perper discussed the role of mixed coal dust containing silica or coal workers' pneumoconiosis in causing centrilobular emphysema in Mr. Sexton. Dr. Perper noted that centrilobular emphysema is a known result of smoking and Mr. Sexton was a significant smoker, though he quit 19 years before he died. However, recently coal dust has been known to cause the disease and studies have shown that emphysema is present more often and is more advanced in patients with coal workers' pneumoconiosis.

Based on the autopsy findings indicative of complicated coal workers' pneumoconiosis with associated centrilobular emphysema and Mr. Sexton's occupational history, Dr. Perper believes that coal workers' pneumoconiosis was a cause of functional pulmonary disability, during his life, and was responsible for the chronic obstructive lung disease, chronic bronchitis and numerous acute exacerbations of such. Although Mr. Sexton developed a unilateral thrombosis of carotid arteries, complicated coal workers' pneumoconiosis was still a contributing cause of Mr. Sexton's death and a hastening factor in his death. Because Mr. Sexton's terminal hospitalization started with severely increased shortness of breath and the thrombosis of the carotid arteries was subsequent to this episode, the complicated coal workers' pneumoconiosis played a role in Mr. Sexton's death. Additionally, the thrombosis of the carotid arteries occurred within vessels free of arteriosclerosis, which indicates it did not occur because of degenerative vascular atherosclerosis but by increased blood coagulability, "a process that might have been induced by steroids given to Mr. Sexton for his pneumonia." Dr. Perper also attributes Mr. Sexton's coal workers' pneumoconiosis to his death due to the hypoxemia, multiple episodes of acute pulmonary infections with a background of complicated coal workers' pneumoconiosis and chronic lung disease. Mr. Sexton did not die suddenly as the result of a stroke; instead, he expired due to severe pulmonary impairment and cardiac arrhythmia, with a cerebral vascular accident markedly aggravated by chronic lung disease. Chronic lung disease results in hypoxemia precipitating/aggravating a cardiac arrhythmia in an individual with heart disease.

Dr. Perper summarized his conclusions: 1) Mr. Sexton had evidence of extensive complicated coal workers' pneumoconiosis associated with centrilobular emphysema; 2) Mr. Sexton's complicated pneumoconiosis resulted from his more than 26 years of exposure to coal dust containing silica, a more than sufficient period to develop coal workers' pneumoconiosis; and, 3) coal workers' pneumoconiosis and associated centrilobular emphysema were contributory causes of Mr. Sexton's death and hastening factors both directly and indirectly through chronic and acute pulmonary insufficiency and hypoxemia, which triggered or aggravated a fatal arrhythmia.

In a deposition taken on July 12, 2004, Dr. Perper elaborated on his conclusions regarding the role of pneumoconiosis in Mr. Sexton's death. Dr. Perper explained that complicated coal workers' pneumoconiosis is present when a nodule of coal workers'

pneumoconiosis exceeds one to two centimeters in size on autopsy, which has changed recently because the old requirement was at least two centimeters but recent sources differ. Dr. Perper reiterated that there was no evidence of tuberculosis on the lung tissue samples. Dr. Perper agreed with Dr. Naeye's deposition testimony that the lesions in Mr. Sexton's lungs are silicotic and that they exceed one centimeter in size. However, in disagreement with Dr. Naeye, Dr. Perper observed lesions up to two centimeters in size in the lung tissue sample with areas of fibro-anthracosis in the pneumoconiotic nodules. Dr. Perper firmly believes that the nodules of pneumoconiosis would show as opacities larger than one centimeter on an x-ray. Although it is difficult to explain why some radiologists did not observe such opacities, Dr. Perper indicated that it is not unusual for radiologists to miss complicated pneumoconiosis. Dr. Perper notes in addition to clinical pneumoconiosis, Mr. Sexton also had legal pneumoconiosis through his chronic bronchitis and centrilobular emphysema diagnoses.

On cross-examination, Dr. Perper explained that he evaluated eight slides of the lung, which he believes allowed him to review a representative portion of the lung. Studies have shown that 30 percent of chest x-ray readings are incorrect, so he believes the x-ray readers interpreted the radiographic studies wrong, calling opacities granuloma or rendering negative interpretations.

Discussion

As Dr. Naeye noted, and implicit in the *Scarboro* analytical model, radiographic images tend to understate the actual size of pulmonary lesions. Considering that radiographic complicated pneumoconiosis lesions are defined as opacities greater than one centimeter, in assessing whether autopsy findings represent "large" lesions, the baseline size is a mass greater than one centimeter. The pathologists who evaluated the post-mortem evidence disagreed on whether the pulmonary masses in Mr. Sexton's lungs exceeded one centimeter. Although Dr. Bush, a board certified pathologist, microscopically observed numerous pulmonary masses, none of the lesions exceeded 0.9 centimeters in diameter. However, the remaining three pathologists, including two with board certification, found pulmonary lesions greater than one centimeter. Upon gross examination, Dr. Coogan identified a two centimeter mass of coalesced anthracosis macules.²² Under the microscope, Dr. Perper discovered pneumoconiosis lesions measuring up to two centimeters, which were displacing lung tissue. During microscopic examination, Dr. Naeye observed masses containing silicotic crystals admixed with black pigment that was coal workers' pneumoconiosis. The masses exceeded one centimeter in size, measuring up to 1.2 centimeters.

In assessing this dimensional professional dispute, I note Dr. Bush's assertion that the other pathologists may have overstated the size of lesions in the lung tissue due to elongation associated with magnification. However, neither Dr. Perper nor Dr. Naeye, the two other board certified pathologists, nor Dr. Coogan expressed any qualification on their size assessments attributable to the processing and examination of the slides. Dr. Bush also challenged Dr. Coogan's gross examination finding of a two centimeter mass based on his own microscopic

²²While Dr. Coogan did not specify the dimensions of the microscopic nodules, her microscopic examination confirm the presence of multiple variably sized densely hyalinized macules which coalesced and were associated with anthrasilicotic pigment deposition consistent with coal workers' pneumoconiosis.

determination that the lung tissue only contained “adjacent” or “near” lesions, each less than 0.9 centimeters in size. Once again, however, when Dr. Coogan, Dr. Naeye, and Dr. Perper evaluated the same lung tissue slides, none of the pathologists felt compelled to alter or challenge Dr. Coogan’s gross autopsy findings. After considering Dr. Bush’s two concerns, I nevertheless find the consensus of Dr. Coogan, Dr. Perper, and Dr. Naeye represents the preponderance of the evidence and establishes that pulmonary lesions greater than one centimeter were present in Mr. Sexton’s lungs.

2. Equivalency to Chest X-Ray Opacity

According to the *Scarboro* court, the benchmark for defining “massive lesion” in an autopsy under 20 C.F.R. § 718.304 (b) is the “objective” chest x-ray standard of an opacity greater than one centimeter. 220 F.3d at 256. Having determined based on the preponderance of the pathologists’ opinions that Mr. Sexton had a large lesion in his lungs at the time of his death, I must next assess whether that lesion was the equivalent of a chest x-ray opacity greater than one centimeter, such that it is considered a “massive” lesion necessary to support a legal finding of complicated pneumoconiosis.

Prior to making that equivalency determination, I return to the pathologists who found a large mass to ascertain the actual dimension of the large pulmonary lesion. In review, on gross examination, Dr. Coogan observed a two centimeter pulmonary mass. Through microscopic evaluation, Dr. Perper determined the largest masses measured two centimeters. Based solely on his microscopic evaluation, Dr. Naeye was unable to definitely state the lesions exceeded 1.2 centimeters due to the limitation of lung tissue sample on the slide.

While Dr. Perper and Dr. Naeye engaged in a dispute on whether the lung tissue sample on the microscope slide established the presence of a mass larger than 1.2 centimeters, neither pathologist disputed Dr. Coogan’s gross examination finding. Additionally, at least in his initial report, Dr. Naeye acknowledged that the lung tissue sample might only contain a portion of a larger lesion upon which a physician could postulate that the size of the masses in Mr. Sexton’s lungs may actually have reached two centimeters. Notably, Dr. Perper specifically identified one lung tissue slide that appeared to contain only have half of a much larger lesion. Consequently, I find the gross examination autopsy findings of Dr. Coogan, as supported by Dr. Perper’s findings and Dr. Naeye’s acknowledgment, establish that the largest pulmonary lesions in Mr. Sexton’s lungs were two centimeters.

Having determined the size of the largest pulmonary mass, I must determine whether a two centimeter mass discovered during the autopsy of Mr. Sexton’s lungs is equivalent to a radiographic opacity greater than one centimeter. Only Dr. Perper and Dr. Naeye addressed the radiographic equivalency issue concerning the large pulmonary mass.²³ According to Dr. Perper, the two centimeter microscopic lesion would show as an opacity larger than one centimeter on chest x-ray. Initially, Dr. Naeye stated that the 1.2 centimeter pneumoconiosis lesion that he observed under the microscope would not show up as a one centimeter radiographic opacity and he disputed the ability to equate a two centimeter autopsy mass to a radiographic opacity greater

²³Since Dr. Bush concluded that the 0.9 centimeter, “adjacent” masses he observed microscopically would not be radiographically significant, he did not address the chest x-ray equivalency of a larger pulmonary mass.

than one centimeter. However, in a subsequent deposition, Dr. Naeye acknowledged the development of a medical consensus that established a two centimeter lesion on autopsy would appear to be “not much more than one centimeter” on a chest x-ray. Finally, I have considered the absence of any actual radiographic evidence of an opacity greater than one centimeter. However, the *Scarboro* court only required an equivalency determination and not actual radiographic evidence of complicated pneumoconiosis. Further, both Dr. Perper and Dr. Naeye indicated that a chest x-ray is a less accurate diagnostic tool than an autopsy. Accordingly, based on the representations of Dr. Perper and Dr. Naeye, I find that the two centimeter lesion in Mr. Sexton’s lungs would appear as an opacity greater than one centimeter on chest x-ray.

Other Medical Evidence

Since Mrs. Sexton has proven the existence of a pulmonary mass in her husband’s lungs equivalent to the requisite chest x-ray large opacity, I move to the second adjudicative step added by the court in *Scarboro* and consider other relevant medical evidence prior to making a determination whether Mrs. Sexton has invoked the 20 C.F.R. § 718.304 presumption. According to the *Scarboro* court, in this second stage of the analysis, I must determine whether the preponderance of the other medical evidence affirmatively shows that the large opacity was caused by some other intervening pathology than coal workers’ pneumoconiosis. In Mr. Sexton’s case, the “other” medical evidence has two components: a) other objective pulmonary test results and b) medical opinion.

Pulmonary Function Tests

Exhibit	Date / Doctor	Age / Height	FEV ¹ pre ²⁴ post ²⁵	FVC pre post	MVV pre post	% FEV ¹ / FVC pre post	Qualified ²⁶ pre Post	Comments
DX 38	Jan. 7, 1985 Coburn Medical Office	45 72"	3.96	4.94	125	80.2%	No ²⁷	Normal
DX 38	Nov. 8, 1999 Dr. T. Banchuin	58 72"	2.83 2.68	3.67 3.40	102 112	77.1% 78.8%	No ²⁸ No	Minimal airway obstruction

²⁴Test result before administration of a bronchodilator.

²⁵Test result following administration of a bronchodilator.

²⁶Under 20 C.F.R. § 718.204 (b) (2) (i), to qualify for total disability based on pulmonary function tests, for a miner’s age and height, the FEV1 must be equal to or less than the value in Appendix B, Table B1 of 20 C.F.R. § 718, **and either** the FVC has to be equal or less than the value in Table B3, or the MVV has to be equal **or** less than the value in Table B5, or the ratio FEV1/FVC has to be equal to or less than 55%.

²⁷ The qualifying FEV1 number is 2.46 for age 45 and 72.0”; the corresponding qualifying FVC and MVV values are 3.08 and 98, respectively.

²⁸The qualifying FEV1 number is 2.25 for age 58 and 72.0”; the corresponding qualifying FVC and MVV values are 2.85 and 90, respectively.

Arterial Blood Gas Studies

Exhibit	Date / Doctor	pCO ² (rest) pCO ² (exercise)	pO ² (rest) pO ² (exercise)	Qualified ²⁹	Comments
DX 38	Nov. 8, 1999 Dr. T. Banchuin	32.8	76.6	No ³⁰	
DX 13	Dec. 10, 1999 Dr. Barongan	29.4	45.3	Yes ³¹	
DX 13	Dec. 10, 1999 Dr. Barongan	35.5	45.8	Yes ³²	

Discussion

Other than the period of his last hospital admission when he struggled with pneumonia and cerebral stroke, Mr. Sexton's pulmonary test results did not demonstrate any significant abnormalities. However, these pulmonary studies do not negate a finding of complicated pneumoconiosis because no physician definitively relied on the results to identify a different etiology for the pulmonary lesions. While both Dr. Naeye and Dr. Bush highlighted the absence of a significant pulmonary impairment in assessing whether pneumoconiosis played a significant contributing role in Mr. Sexton's death, neither doctor asserted the pulmonary test results pointed to some other cause for the anthracosis nodules. Additionally, standing alone, Mr. Sexton's pulmonary function tests and blood gas studies, which do not specifically isolate the cause of a pulmonary impairment, do not provide affirmative evidence that the large opacities in his lungs are due to some other pathology. Consequently, I find the pulmonary function and arterial blood gas tests do not establish some cause other than coal dust inhalation for the two centimeter pulmonary mass.

Medical Opinions

June 1980 Hospital Records DX 41

From June 12, 1980 to June 22, 1980, Mr. Sexton was in the hospital after a coal mine injury. The physician reported Mr. Sexton's smoking history of one pack of cigarettes a day for 15 years. A left pleural abrasion was performed to fix the pneumothorax.

On June 15, 1980, Dr. L. Lerschlolarn diagnosed a pneumothorax when Mr. Sexton presented with acute chest pain. A chest exam revealed diminished breath sounds. The arterial

²⁹To qualify for Federal Black Lung Disability benefits at a coal miner's given pCO² level, the value of the coal miner's pO² must be equal to or less than corresponding pO² value listed in the Blood Gas Tables in Appendix C for 20 C.F.R. § 718.

³⁰ For the pCO² of 32, the qualifying pO² is 68, or less.

³¹For the pCO² of 29, the qualifying pO² is 71, or less.

³² For the pCO² of 35, the qualifying pO² is 65, or less.

blood gas study showed hypoxemia and hyperventilation. The chest x-ray showed Mr. Sexton's collapsed lung.

TB Test
DX 38

On May 14, 1984, Dr. John G. Byers conducted a tuberculosis test, which produced strongly positive results. The chest x-ray indicated lesions of tuberculosis and new bi-apical lesions.

DX 41

On May 20, 1984, Mr. Sexton tested positive for tuberculosis. A final diagnosis for Mr. Sexton was bilateral and apical infiltrates and possible tuberculosis.

Dr. Robert Strang
DX 44

On February 10, 1986, Dr. Strang noted in a hospital treatment record that Mr. Sexton had a history of old chest condition including tuberculosis.

December 1999 Hospital Admission
DX 13, DX 38

On December 8, 1999, Mr. Sexton was admitted to the hospital. Dr. Barongan diagnosed right lower lobe pneumonia, exacerbation of chronic obstructive lung disease and cerebrovascular accident plus probably brain stem and cardiac dysrhythmia. Dr. Barongan noted a history of tuberculosis, onset of progressive dyspnea, and productive cough. Mr. Sexton had a history of COPD and diminished breath sounds were noted.

On that day, Dr. Charles Scott Pullen diagnosed pneumonia. A chest exam revealed lungs with decreased breath sounds and mild crackles.

Dr. Coogan

Concerning the two centimeter coalesced macules, Dr. Coogan found evidence of anthracosis, consistent with coal workers' pneumoconiosis. Dr. Coogan also noted there was no evidence of tuberculosis or cancer. Based on her gross and microscopic examinations, Dr. Coogan diagnosed as pulmonary ailments bilateral anthrasilicosis compatible with coal workers' pneumoconiosis, severe left pulmonary adhesion, bacterial infection with cavity scarring, and bronchopneumonia.

Dr. Naeye

In several of the masses exceeding one centimeter, Dr. Naeye found silicotic crystals and diagnosed pneumoconiosis in addition to fungal infection cavities and pneumonia. Additionally,

no evidence of tuberculosis was present. Yet, for three principal reasons, Dr. Naeye opined that Mr. Sexton did not have complicated pneumoconiosis. First, the masses in Mr. Sexton's lungs were not consistent with "classic" complicated pneumoconiosis, which is an immunological disorder. Specifically, the lesions did not contain "black fluid" which is typically present due to the destruction of lung tissue by the inflammatory process of complicated pneumoconiosis. Second, Mr. Sexton's pulmonary lesions had been stable for some time; whereas, complicated pneumoconiosis is a rapidly advancing pulmonary disease. Third, complicated pneumoconiosis predisposes a person to pneumonia; yet, Mr. Sexton had normal pulmonary function and worked up until the time of his death.

Dr. Bush

Dr. Bush diagnosed coal workers' pneumoconiosis but concluded complicated pneumoconiosis was not present. Complicated coal workers' pneumoconiosis causes heavy black pigmentation on the lungs' surfaces and in cross sections. In Mr. Sexton's case, other than the lesions in the apices, the surfaces and cross-sections of his lungs were only mildly pigmented red-yellow. Additionally, complicated pneumoconiosis represents progressive and massive fibrosis. Mr. Sexton's lesions were neither. The pulmonary masses had been stable for years and were not advancing or progressing. The clusters of pulmonary masses existed only in the apices of the lungs and were too limited to be characterized as "massive."

Dr. Perper

After finding no evidence of tuberculosis, Dr. Perper concluded the "massive" pulmonary nodules found during Mr. Sexton's autopsy established the presence of complicated coal workers' pneumoconiosis. To support his diagnosis, he emphasized the magnitude of the masses, their progression, and the deterioration of Mr. Sexton's pulmonary function.

Discussion

Prior to his death, Mr. Sexton experienced two pulmonary issues. First, in 1980, he suffered a collapsed left lung and underwent a procedure to adhere a portion of the lung to the pleura to preclude further collapse. During the autopsy, Dr. Coogan noted the left lung adhesions. However, that finding was unrelated to the pulmonary nodules.

Second, in May 1984, Mr. Sexton tested positive for tuberculosis and the physicians found lesions of tuberculosis present on a chest x-ray. A few days later, in the physician's final diagnosis, he noted "possible tuberculosis." However, other procedures failed to substantiate its presence and according to Dr. Coogan, Dr. Naeye, and Dr. Perper, no evidence of tuberculosis was found during the autopsy. Consequently, tuberculosis is not a viable etiology for the pulmonary mass.

During the autopsy, Dr. Coogan identified a pulmonary cavity associated with a bacterial infection which she diagnosed separately from the coal workers' pneumoconiosis. Dr. Perper did not discuss the bacterial infection. Though Dr. Naeye and Dr. Bush noted the presence of a pulmonary cavity due to the bacterial/fungal infection, neither pathologist attributed the

pulmonary lesions solely to the bacteria or fungus; significantly, they also found evidence of anthracosis and silicotic crystals in the lesions.

Next, although Dr. Coogan discovered a two centimeter, anthracotic mass during gross examination of the lungs, she did not specifically diagnose complicated pneumoconiosis. However, her failure to include complicated pneumoconiosis as a diagnosis and silence on the issue does not represent affirmative evidence that the large opacities were caused by some other intervening pathology, especially since the doctor clearly associated the pulmonary masses with anthracosis.

The remaining three pathologists who evaluated the post-mortem evidence had contrary opinions on whether Mr. Sexton had complicated pneumoconiosis. Dr. Perper diagnosed complicated pneumoconiosis. Dr. Naeye and Dr. Perper did not. Due to this professional agreement, I must assess the relative probative weight of their opinions on the basis of documentation and reasoning.

As to the first factor, a physician's medical opinion is likely to be more comprehensive and probative if it is based on extensive objective medical documentation such as radiographic tests and physical examinations. *Hoffman v. B & G Construction Co.*, 8 B.L.R. 1-65 (1985). In other words, a doctor who considers an array of medical documentation that is both long (involving comprehensive testing) and deep (includes both the most recent medical information and past medical tests) is in a better position to present a more probative assessment than the physician who bases a diagnosis on a test or two and one encounter.

The second factor affecting relative probative value, reasoning, involves an evaluation of the connections a physician makes based on the documentation before him or her. A doctor's reasoning that is both supported by objective medical tests and consistent with all the documentation in the record, is entitled to greater probative weight. *Fields v. Island Creek Coal Co.*, 10 B.L.R. 1-19 (1987). Additionally, to be considered well reasoned, the physician's conclusion must be stated without equivocation or vagueness. *Justice v. Island Creek Coal Co.*, 11 B.L.R. 1-91 (1988).

With these principles in mind, and noting all three pathologists had a firm documentary foundation, I conclude that the opinions of Dr. Naeye and Dr. Bush that Mr. Sexton did not have complicated pneumoconiosis have diminished relative probative value due to reasoning deficiencies, in legal terms.

Dr. Naeye's medical opinion has diminished probative value as contrary evidence of complicated pneumoconiosis due to his apparent disagreement with the legal standard for complicated pneumoconiosis. After acknowledging the legal definition of complicated pneumoconiosis, Dr. Naeye nevertheless challenges that definition by concluding Mr. Sexton does not have complicated pneumoconiosis since other typical medical characteristics of the disease are absent. While Dr. Naeye's analysis may be reasonable in medical terms, the regulation, as interpreted by the *Scarboro* court, defines complicated pneumoconiosis as "one or more large opacities" established by radiographic evidence, or its equivalent (emphasis added).³³

³³20 C.F.R. § 718.304 (a).

The legal standard does not require additional medical characteristics mandated by Dr. Naeye of “black fluid,” rapid progressivity, massive perfusion, or total respiratory disability to establish the presence of complicated pneumoconiosis. Notably, in determining that Mr. Sexton did not have complicated pneumoconiosis, Dr. Naeye does not really challenge the etiology of the pulmonary masses. He concludes Mr. Sexton’s exposure to coal mine dust was at least one of the causes for the pulmonary masses. In his opinion, the large pulmonary nodules containing silica are consistent with of coal workers’ pneumoconiosis.

Dr. Bush’s analysis in determining that Mr. Sexton did not have complicated pneumoconiosis also conflicts with the legal definition and consequently has diminished probative value. Like Dr. Naeye, Dr. Bush stresses the absence of classic medical symptoms of complicated pneumoconiosis in determining that Mr. Sexton did not have the disease. Yet, in reaching that conclusion, Dr. Bush does not offer a different etiology for the pulmonary lesions. Instead, in concurrence with the other pathologists, Dr. Bush opined that some of the pulmonary “clusters” and scarring in Mr. Sexton’s lungs were consistent with coal workers’ pneumoconiosis and silicosis.

In contrast, Dr. Perper’s opinion is reasoned within the parameters of the legal standard. Additionally, his diagnosis of complicated pneumoconiosis supports a determination that coal dust, rather than some other etiology, has caused the large lesion in Mr. Sexton’s lungs.

In summary, according to the *Scarbro* court, once the presence of large opacities as defined by the Act is established, the invocation of the presumption under 20 C.F.R. § 718.304 is precluded only if an affirmative showing is made that some pathology, unrelated to exposure to coal dust, had caused the lung masses to develop. 220 F.3d at 250. The other objective medical evidence in the record fails to provide sufficient affirmative showing that some etiology, other than coal dust, caused the large pulmonary mass. Accordingly, I find Mr. Sexton had complicated pneumoconiosis.

Conclusion

Mrs. Sexton is able to invoke the irrebuttable presumption that Mr. Sexton’s death was due to pneumoconiosis under 20 C.F.R. § 718.304 through a) the presence of a large pulmonary nodule in Mr. Sexton’s lungs identified upon autopsy, which is the equivalent of a chest x-ray opacity greater than one centimeter; and b) the absence of other medical evidence that affirmatively establishes another cause for the pulmonary mass unrelated to coal dust exposure.³⁴

Issue # 2 – Entitlement to Benefits

My finding that Mrs. Sexton established that her husband had complicated pneumoconiosis and the corresponding invocation of the irrebuttable presumption under 20 C.F.R. § 718.304 that his death was due to pneumoconiosis represents a mistake in determination of fact by the Benefits Review Board in their denial of her claim, which warrants modification of

³⁴Since the presence of complicated pneumoconiosis invokes an irrebuttable presumption of death due to coal workers’ pneumoconiosis under 20 C.F.R. § 718.304, I need not address the other means available to establish death due to black lung disease.

that denial. As a result, I must next determine whether Mrs. Sexton is entitled to survivor benefits is necessary.

As previously discussed, to receive survivor benefits under the Act, Mrs. Sexton must prove that: 1) she is an eligible survivor of the deceased miner; 2) that Mr. Sexton had pneumoconiosis; 3) that his pneumoconiosis arose out of coal mine employment; and, 4) Mr. Sexton's death was due to pneumoconiosis. Based on the parties' stipulation, Mrs. Sexton has established her eligibility as a surviving spouse. Through her invocation of the irrebuttable presumption that Mr. Sexton's death was due to pneumoconiosis, Mrs. Sexton has also established that Mr. Sexton had pneumoconiosis and that his death was due to pneumoconiosis. Consequently, to receive benefits, Mrs. Sexton must establish that Mr. Sexton's pneumoconiosis arose out of his coal mine employment.

Pneumoconiosis Arising Out of Coal Mine Employment

The parties have stipulated that Mr. Sexton had at least twenty-six years of coal mine employment. As indicated earlier, under the regulations, if a miner works ten or more years in one or more mines, a presumption exists that his pneumoconiosis arose out of coal mine employment.

Because the presumption of pneumoconiosis arising out of coal mine employment is rebuttable, I must reexamine the medical record to determine whether sufficient evidence exists to sever the presumptive connection between Mr. Sexton's pneumoconiosis and his coal mine employment. I have already determined that the medical evidence in this case does not establish a non-coal dust related pathology for the pneumoconiosis discovered in Mr. Sexton's lungs during the autopsy. Accordingly, the causation presumption under 20 C.F.R. § 718.203 (b) has not been rebutted. Consequently, I find Mr. Sexton's pneumoconiosis arose out of his coal mine employment.

CONCLUSION

Based on an evaluation of the entire evidentiary record in conjunction with a consideration of the newly submitted medical opinions, I have determined a mistake in determination of fact occurred in the prior denial of Mrs. Sexton's survivor claim. Through probative autopsy evidence and a radiographic equivalency determination, and in the absence of affirmative contrary etiology evidence, Mrs. Sexton has invoked the irrebuttable presumption under 20 C.F.R. § 718.304 that her husband's death was due to pneumoconiosis. Based on the entire evidence in the record, Mrs. Sexton has also proven that Mr. Sexton's pneumoconiosis arose out of coal mine employment and that she is an eligible survivor under the Act. Accordingly, Mrs. Sexton's claim for survivor benefits under the Act must be approved.

DATE OF ENTITLEMENT

In the case of a coal miner who died due to coal workers' pneumoconiosis, benefits to the survivor are payable beginning the month the coal miner died. 20 C.F.R. § 725.503 (c). Since

Mr. James Sexton passed away on December 11, 1999, Mrs. Molly Sexton's survivor benefits are payable beginning December 1, 1999.

ATTORNEY FEES

Counsel for the Claimant has thirty calendar days from receipt of this decision and order to submit an application for attorney fees in accordance with 20 C.F.R. §§ 725.365 and 725.366. With the application, counsel must attach a document showing service of the fee application upon all parties, including the Claimant. The other parties have fifteen calendar days from receipt of the fee application to file an objection to the request. Absent an approved application, no fee may be charged for representation services associated with this claim.

ORDER

The claim of MRS. MOLLY SEXTON for survivor benefits under the Act is **GRANTED**. The Employer, PARAMOUNT COAL COMPANY, is ordered to:

1. Pay the Claimant, MRS. MOLLY SEXTON, all survivor benefits to which she is entitled under the Act and Regulations. Benefits shall commence December 1, 1999.
2. Reimburse the Black Lung Disability Trust Fund, pursuant to 20 C.F.R. § 725.602, for all interim payments made by the Black Lung Disability Trust Fund to MRS. MOLLY SEXTON;
3. Deduct, as appropriate, from the payments ordered in paragraph one, the amounts reimbursed to the Black Lung Disability Trust Fund as directed in paragraph two; and,
4. Pay to the Secretary of Labor interest as required pursuant to 20 C.F.R. § 725.608 (b).

SO ORDERED:

A

RICHARD T. STANSELL-GAMM
Administrative Law Judge

Date Signed: July 22, 2005
Washington, DC

NOTICE OF APPEAL RIGHTS: Pursuant to 20 C.F.R. § 725.481, any party dissatisfied with this Decision and Order may appeal it to the Benefits Review Board within 30 days from the date this decision is filed with the District Director, Office of Worker's Compensation Programs, by filing a notice of appeal with the Benefits Review Board, ATTN.: Clerk of the Board, Post Office Box 37601, Washington, DC 20013-7601. See 20 C.F.R. § 725.478 and § 725.479. A copy of a notice of appeal must also be served on Donald S. Shire, Esquire, Associate Solicitor for Black Lung Benefits. His address is Frances Perkins Building, Room N-2117, 200 Constitution Avenue, NW, Washington, DC 20210.